Mol Cell Biol. 2004 Jan;24(1):96-104.

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Essential role for ADAM19 in cardiovascular morphogenesis.

Zhou HM, Weskamp G, Chesneau V, Sahin U, Vortkamp A, Horiuchi K, Chiusaroli R, Hahn R, Wilkes D, Fisher P, Baron R, Manova K, Basson CT, Hempstead B, Blobel CP.

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Congenital heart disease is the most common form of human birth defects, yet much remains to be learned about its underlying causes. Here we report that mice lacking functional ADAM19 (mnemonic for a disintegrin and metalloprotease 19) exhibit severe defects in cardiac morphogenesis, including a ventricular septal defect (VSD), abnormal formation of the aortic and pulmonic valves, leading to valvular stenosis, and abnormalities of the cardiac vasculature. During mouse development, ADAM19 is highly expressed in the conotruncus and the endocardial cushion, structures that give rise to the affected heart valves and the membranous ventricular septum. ADAM19 is also highly expressed in osteoblast-like cells in the bone, yet it does not appear to be essential for bone growth and skeletal development. Most adam19(-/-) animals die perinatally, likely as a result of their cardiac defects. These findings raise the possibility that mutations in ADAM19 may contribute to human congenital heart valve and septal defects.

PMID: 14673146 [PubMed - indexed for MEDLINE]